more, potent inhibition of the target by aromatic sulphonamides is well understood, allowing the rational design of such carrier sulphonamides (*5* and references therein). In vitro incubation of whole human blood with radioiodinated pIBS gives quantitative uptake by red cells in less than 1 min at 37°C. In vivo administration of the radiopharmaceutical, involving a single intravenous injection, is just as effective, and it clears from the blood at least eight times more slowly than the standard technetium-red cell label. Consequently, blood-pool images can be obtained using far less activity and over longer periods than are routinely employed for ^{99m}Tc; moreover, the problems that are now occasionally encountered with patients on medication are unlikely to arise when using this selective approach.

While ^{123}I has excellent imaging properties and [^{123}I]pIBS is amenable to kit preparation, the cost and limited availability of the radionuclide vitiates its use in routine blood cell labeling. However, a combination of our approach with the use of ^{99m}Tc would present an ideal short-term blood-pool imaging agent. We recently have been investigating ^{99m}Tc-propyleneamineoxime derivatized sulphonamides in this regard (*6*), and other preliminary studies in this area have been reported by Subramanian and coworkers (*7*). Unfortunately our results have not yet matched those with radioiodinated pIBS, since there is significant plasma binding of the technetium complexes; however, an equally efficacious agent should eventually emerge.

The simplicity of the protocol, the selectivity of the carrier, and its persistent retention suggests that the enzyme-inhibitor approach to red cell labeling will not only be of value to short-term studies but could also offer significant advantages to longer term studies such as the determination of in vivo red cell survival.

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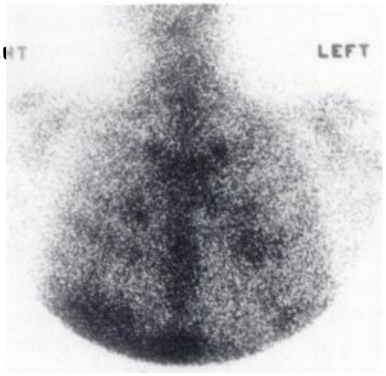

FIGURE 1. Anterior ^{67}Ga-citrate scan showing increased lung uptake and absence of deltoid muscle uptake.

Gallium Uptake in Eosinophilia Myalgia Syndrome

TO THE EDITOR: In a recent case report on gallium uptake in eosinophilia myalgia syndrome (EMS), Kim et al. (*1*) reported gallium lung uptake but absence of uptake in the muscle. The authors attributed the lack of muscle uptake to prior steroid therapy. We wish to report a patient with EMS, who had positive muscle biopsy but failed to take up gallium at that site even though he was not on steroid therapy.

A 64-yr-old male presented with fatigue, myalgia, muscle cramps, and swelling of his extremities. Examination showed non-pitting edema in the extremities and moribiliform rash on the abdomen. He had been taking L-tryptophan for 6 mo for insomnia. Laboratory data revealed increased sedimentation rate and absolute eosinophilia of 1170/mm^3. Chest x-ray demonstrated bi-basilar reticular infiltrates. A gallium lung scan showed bilateral basal uptake and normal uptake in the deltoid muscles (Fig. 1).

Muscle biopsies from both deltoid areas (including skin and fascia) were compatible with eosinophilic fascitis and EMS. Open lung biopsy showed diffuse interstitial fibrosis (desquamative interstitial pneumonia). This case points to the nonspecificity of gallium lung uptake and the need to pursue tissue diagnosis. Absence of gallium muscle uptake, as seen in this case, may be a feature of this disorder.

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REPLY: We wish to thank Dr. Rao et al. for their comments on our recent paper (*1*).

Before responding to their letter, we must note that we had difficulty in understanding their description of Figure 1, which they described as "bilateral basal uptake," though the image shown does not have increased uptake at the bases. In addition,

in the legend, they describe "increased lung uptake". Presumably, they imply diffuse lung uptake, but there appears to be increased uptake in the perihilar regions bilaterally which may be at the costochondral junctions rather than hilar or lung uptake.

We have stated that the absence of ^{67}Ga uptake in muscular distribution in our case may be related to prior steroid therapy. However, other factors such as predominant cellular infiltration by lymphocytes and plasma cells, but rarely by eosinophils on muscle biopsy, may be another reason for the lack of ^{67}Ga uptake (*2*).

We agree with the authors, that gallium lung uptake in eosinophilia myalgia syndrome (EMS) is a nonspecific finding. However, we believe the gallium scan could be used in defining the disease process and in monitoring the response to the treatment.

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Proposal of a Modified Scintigraphic Method to Evaluate Duodenogastroesophageal Reflux

TO THE EDITOR: We have read with interest the article of Borsato et al. (*1*) and concur with the idea that not only the severity of the reflux but also the duration of the reflux episode should be evaluated. An index taking into account the amount of refluxed ^{99m}Tc HIDA into the stomach multiplied by the duration of the reflux may offer an interesting parameter.

However, the question arises that there is no correlation between scintigraphic grading and the presence of alkaline exposure on pH monitoring (*1*) and if there is no correlation between the intensity of the reflux and the endoscopic findings (*2*), then why do we need an index? Is it not enough just to detect the reflux?

There are multiple causes for gastritis (*3*). Duodenogastric reflux, although not accepted by everybody (*4*), is one of them, but the endoscopic finding of damaged gastric mucosa does not give a clue about the origin of the damage. Therefore, we wonder if it is possible to find a correlation between the endoscopic findings and the detection of alkaline reflux, either by pH monitoring or by scintigraphic duodenogastric reflux. Let us review the data of Borsato et al. (*1*) in which there are 7 of 25 patients (28%) with gastritis, but in these 7 patients the scintigraphic findings as well as the pH monitoring were negative. Was this a gastritis due to alkaline reflux or to another etiology? Could the lack of correlation be due to differences in etiology of gastritis?

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REPLY: We appreciate the opportunity to reply to Dr. Roland's letter concerning our paper. Indeed, about one-third of our patients (28%) had gastritis and no evidence of pathologic duodenogastric reflux on scintigraphy and pH monitoring. Whether this reflects true absence of reflux or inaccuracy of the current tests is hard to know.

We agree with Dr. Roland that differences in the etiology of gastritis may explain the negative findings with the tests for reflux. As it was pointed out in the discussion of our paper, factors other than reflux, such as *Helicobacter pylori* infection, should be considered in the pathogenesis of antral gastritis (*1*).

Another problem may be the low dependability of the currently available tests in the detection of an increased frequency of sporadic reflux events (*2*). Quantitation of duodenogastric reflux is a formidable task, and at present there is no single test that can be used with confidence to assist in the choice of treatment for the individual patient (*3*).

Although the concerns raised by Dr. Roland are reasonable, we believe that further validation and development of the diagnostic techniques may help in understanding pathophysiology and in providing better management for symptomatic patients.

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Detection of Reversible Thallium-201 Defects With Ribose

TO THE EDITOR: In the February 1991 issue of *The Journal of Nuclear Medicine*, our paper showing that ribose increased the detection of reversible ^{201}Tl defects (*1*) was followed by an editorial (*2*) which raised the following general issues.

The editorial referred to the differences in the number of "reversible defects" observed at 1 hr and at 4 hr delayed imaging.